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Reply to: Co-existence of congestion and preload-dependence identified by pulse pressure respiratory variations: right ventricular afterload might be the key

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To the editor,

We appreciate the thoughtful comments by Bussy et al. [1] regarding our study on the coexistence of fluid responsiveness (FR) and venous congestion (VC) in critically ill patients [2]. We agree that the intersection between fluid responsiveness, right ventricular (RV) dynamics, and fluid tolerance is complex and warrants ongoing discussion and investigation. However, we would like to address several points raised in the letter, many of which stem from inferences rather than actual data presented.

First, regarding the use of pulse pressure variation (PPV) to assess FR: we applied PPV and other tests in accordance with expert recommendations [3, 4], using a pragmatic approach reflective of real-world ICU practice as we did in our prior studies [5]. Physicians selected the most appropriate test based on individual clinical context (e.g., arrhythmias, spontaneous breathing), and a second confirmatory test was performed in 11% of cases when uncertainty arose. While we acknowledge the limitations of PPV in certain clinical states—including altered lung compliance or RV dysfunction—we carefully considered its validity criteria. The critique regarding presumed use of low tidal volumes (< 8 mL/kg PBW) is speculative, as this was not systematically the case in our cohort. In fact, following current research, we transiently increased tidal volumes above 8 mL/kg PBW to validate PPV when needed [6]. Therefore, concerns about widespread misclassification of FR based on this assumption are likely overstated.

Importantly, preload responsiveness is dynamic during critical illness. As we have previously demonstrated, many FR+ patients evolve into an FR− state over short periods of time [5]. Similar patterns were reported in the FRESH trial, where fluid responsiveness decreased as time from inclusion progressed [7]. A more plausible explanation for the 38% FR+ rate in our cohort is the broad inclusion window (up to 24 h post-ICU admission); many patients were likely assessed later in their resuscitation trajectory, having already transitioned to a FR− state [5]. Indeed, the median time to study measurement was 7 [1–16] hours after ICU admission.

Second, Bussy et al. correctly suggest that RV afterload effects may mimic FR+ status on PPV and that could explain why patients had a positive FR+ while congestion signals were present. While physiologically sound, this hypothesis is not well supported by our data. Only 23% (21) of patients had RV/LV area ratios > 0.6, and only five of them were categorized as FR+. Among these 21 patients, none had RV/LV area ratios of > 1, the median

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CVP was 8 [6–11.8] mmHg and TAPSE was 19 [15–25] mm, suggesting preserved RV function. Additionally, this subgroup had a mean PEEP of 8 [5–8] cmH_2O, driving pressure of 11 [10–16] cmH_2O, PaO_2/FiO_2 ratio of 186 [110–302], and $PaCO_2$ of 39 [36–46] mmHg—all of which argue against marked RV afterload. Furthermore, in 38% (8) of these cases, FR was determined using passive leg raising (PLR), a technique not influenced by heart-lung interactions.

We appreciate the authors' call for more invasive and standardized methods to assess preload dependence and VC. While ideal for mechanistic studies, our goal was not to establish a gold standard but rather to explore a pragmatic bedside framework. Notably, other studies using different methodologies have yielded similar findings. For example, Joseph et al. found that using a passive leg raising (PLR) maneuver to assess preload and central venous pressure (CVP) to assess congestion (with a cut-off > 12 mmHg), the prevalence of VC exceeded 25% across FR states. This rate increased above 50% when using a CVP threshold of 8 mmHg [8]. Similarly, Kenny et al., using carotid corrected flow time and internal jugular Doppler during a PLR maneuver, showed heterogeneous responses in both stroke volume and venous congestion signals [9].

Lastly, regarding the VC markers chosen in our study, we prioritized bedside tools that align with standard clinical practice and discussed their limitations transparently in the original manuscript [2]. However, we respectfully disagree with the notion that only pressure-based parameters should be considered the only valid indicators of venous congestion. Doppler-derived indices—such as those in the VExUS score or the renal vein stasis index—have demonstrated a stronger prognostic association to adverse outcomes than pressure-based measures alone in various clinical contexts [10, 11]. But most importantly, integrating these signals synergically rather than competitively could provide a better understanding of such a complex phenomenon. In this line, Guinot et al. employed an unsupervised machine learning approach to define distinct hemodynamic congestion endotypes, including a comprehensive set of variables, such as echocardiography, abdominal organ venous doppler, stroke volume response to passive leg raising, and CVP, among others. They identified 3 distinct endotypes (i.e., hemodynamic congestion, volume overload congestion and systemic congestion), which had different clinical patterns, risks of organ dysfunction and overall clinical trajectories [12]. Thus, integrating signals that provide complementary information regarding the hemodynamic status of the individual patient could allow for further refinement of risk stratification and personalization of therapy, however, further research is still needed.

We are grateful for the opportunity to clarify these points and view this exchange as a constructive step toward improving our collective understanding of hemodynamics in the critically ill.

Abbreviations

FR	Fluid responsiveness
VC	Venous congestion
RV	Right ventricle
PPV	Pulse pressure variation
ICU	Intensive care unit
LV	Left ventricle
TAPSE	Tricuspid Annular Plane Systolic Excursion
PEEP	Positive end expiratory pressure
PLR	Passive leg raising
VExUS	Venous excess ultrasound score

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Data availability
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Declarations

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Not applicable.

Consent for publication
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Competing interests
The authors declare no competing interests.

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